

CASE REPORTS

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Coccidioidomycosis Misdiagnosed as Contact Dermatitis

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ERYTHEMA NODOSUM AND erythema multiforme are hypersensitivity reactions widely known to occur after infection with *Coccidioides immitis*, and these appear in about 20 percent of symptomatic cases.¹ These cutaneous syndromes occur in many other diseases as well and invite differential diagnostic possibilities.² There is little appreciation, however, of another cutaneous lesion of acute coccidioidomycosis which does not denote specific sensitivity to the fungal agent and which, when it occurs, does so at the outset of symptoms rather than two or three weeks later as is the case with erythema nodosum and erythema multiforme. This early lesion has been described as a generalized, erythematous, fine macular eruption simulating "toxic erythemas" of other acute febrile illnesses. Occasionally, it is urticarial or morbilliform.¹

The purpose of this report is to alert physicians to the possibility of coccidioidomycosis in patients from endemic areas who present with

influenza-like symptoms and co-existing rash. The rash may even be the chief complaint, as discussed below.

One of the largest outbreaks of coccidioidomycosis ever reported from a common point source and confirmed by soil cultures occurred in July, 1970, in Northern California.³ The outbreak occurred among 103 archaeology students digging for Indian artifacts 9 miles northeast of Chico in the foothills of the Sierra range. This site was 70 miles northeast of previously documented northern limits of endemicity for coccidioidomycosis. Sixty-one persons developed an illness clinically compatible with coccidioidomycosis, and rash occurred in 30 of them or 49 percent). A previous report estimated the occurrence of rash in patients ill with coccidioidomycosis at 10 percent.¹ Except for two persons with "poison oak," all who had rash also had other symptoms of coccidioidomycosis, such as cough or chest pain. The California State Department of Public Health and other agencies collaborated in the investigation of the outbreak.³

Case histories of two persons are of special interest because both were initially thought to have contact dermatitis but were found by laboratory studies to have coccidioidomycosis.

Reports of Cases

Case 1. A 19-year-old white male student from New York City, who had never before visited areas endemic for coccidioidomycosis, arrived in Chico, California, June 14, 1970. On June 17, he began excavating for Indian ruins and on July 2 began to complain of a generalized, pruritic, urticarial, maculopapular eruption. On July 6 he presented at the college infirmary, where "urticaria" was diagnosed. An antihistamine and topical corticosteroids were prescribed. As symptoms did not improve, he consulted a private physician on July 11 and was admitted to hospital that day. Only cutaneous symptoms were reported in the admission history. Physical examination was

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reported to be unremarkable except for temperature of 38.6°C (101.6°F) and a diffuse, generalized, confluent rash of varying intensity.

The hematocrit was 50 percent and leukocytes numbered 19,000 per cu mm (differential count: 11 percent bands, 74 percent segmented cells, 11 percent lymphocytes, 2 percent monocytes, and 2 percent eosinophils). Blood culture was sterile. Urinalysis was unremarkable except for 4 to 5 white blood cells per high power field. Urine was sterile on culture. No chest x-ray film was taken.

The nurses' notes recorded other complaints: "He aches, has chills, and feels weak." The admitting diagnosis was "allergic dermatitis of unknown etiology," and he was treated with epinephrine, an antihistamine (Benadryl®), calamine lotion, and prednisone 5 mg four times a day. He was discharged on July 15 with a diagnosis of "contact dermatitis." Outpatient therapy of dexamethasone (Decadron®), one 0.75 mg tablet three times a day, was provided.

In a mass survey³ conducted 15 days after onset of his rash, this patient indicated on a questionnaire additional symptoms of shaking chills, malaise, and chest pain. On examination a generalized, confluent, macular rash and circumoral pallor were noted. The patient had a coccidioidin skin test reaction of 17 mm of induration and positive precipitin reaction with *C. immitis* antigen. A posterior-anterior chest x-ray film revealed bilateral lower lobe pneumonia (Figure 1). The patient was lost to follow-up on his return to New York City.

Case 2. A 21-year-old white co-ed from New York who also had never been in areas endemic for coccidioidomycosis arrived in Chico, California, on June 14, 1970. She began digging at the excavation site one day later. On July 11 she became ill, and on July 13 was put into hospital by a private physician. Her admitting history reported a generalized, confluent pruritic rash and "some little discomfort in her chest." On physical examination she had a temperature of 37.8°C (100°F) and a rash which was quite raised in areas. She appeared healthy otherwise.

The admitting diagnosis was "contact dermatitis . . . due to something [she is] doing in school." Laboratory data included a hematocrit of 40 percent and leukocyte count of 13,900 (differential count: 70 percent segmented forms, 3 percent bands, 21 percent lymphocytes, 4 per-

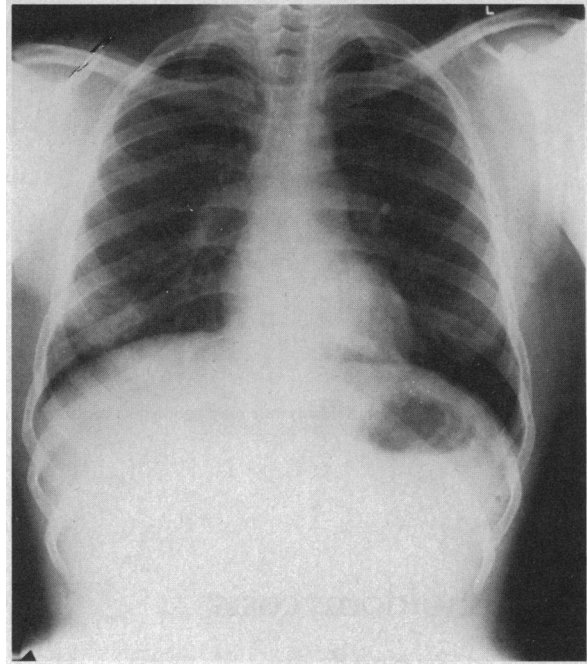


Figure 1.—(Case 1) 19-year-old white male with bilateral lower lobe pneumonia.

cent monocytes, and 2 percent eosinophils). A urinalysis was negative except for 5 to 7 white cells per high power field. No chest x-ray film was taken.

Therapy as an inpatient included epinephrine, dexamethasone (Decadron®), an antihistamine (Benadryl®), and starch baths. The patient was discharged July 15 with Benadryl and Decadron, one 0.75 mg tablet three times a day.

At the mass survey conducted on July 17, she reported that she began having chest pains and shortness of breath on July 8, and on July 11 developed fever, shaking chills, headache, myalgia, stiff neck, night sweats, cough, and the rash described earlier. A coccidioidin skin test was positive at 11 mm of induration and a chest x-ray film showed patchy right lower lobe pneumonia (Figure 2). The patient recovered fully. Her initial symptoms lasted about one week and there was a "slight relapse of chest pains and shortness of breath for a few days in September." She reported that she had been "fine" since then.

Discussion

Both cases illustrate the need to consider coccidioidomycosis in the differential diagnosis of respiratory illness and co-existing rash in persons from endemic areas especially when there is

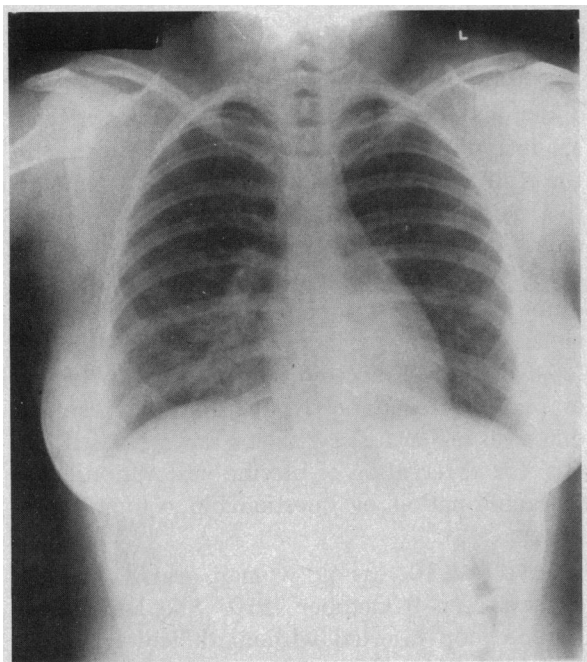


Figure 2.—(Case 2) 21-year-old white woman with patchy right lower lobe pneumonia.

heavy exposure to soil. Even the casual exposure of driving through highly endemic areas such as the San Joaquin Valley has been sufficient for infection by *C. immitis*.⁴ The Chico area was not previously known to be endemic for *C. immitis* and this probably influenced the diagnostic considerations of attending physicians. Until this outbreak, the most northern area in California and the United States documented to be endemic for *C. immitis* was 70 miles southwest of Chico near Brooks, California.⁵

Both patients reported upon here were treated with systemic corticosteroids. Caution should be exercised in the use of such therapy for coccidioidomycosis, since subsequent fungal dissemination has been reported.⁶ On the other hand, Levan and Einstein did treat 19 cases of Valley Fever with a short course of cortisone without evidence of dissemination.⁷ However, persons with "Valley Fever" (that is, primary coccidioidomycosis *with* hypersensitivity reactions of erythema nodosum and/or erythema multiforme) may have a more favorable prognosis with regard to dissemination than those without such hypersensitivity reactions. Indeed, Faber, Smith and Dickson reported that only one in seven hundred with Valley Fever undergo spontaneous dissemination⁸ whereas in symptomatic primary

coccidioidomycosis this complication occurs in about 1 percent of white males and in 12 percent of Negro males.⁹

Summary

Nonspecific "toxic erythema" is not widely appreciated as a concurrent clinical feature of early primary coccidioidomycosis. Two cases are presented in which cutaneous manifestations were, in fact, the chief complaint. Both patients were admitted to hospital with diagnosis of "contact dermatitis" and were treated with systemic corticosteroids. The attendant risks are mentioned. The existence of a generalized, pruritic, maculopapular eruption in patients with influenza-like symptoms coming from endemic areas should alert physicians to the possibility of coccidioidomycosis.

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